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Effect of a high intensity quadriceps fatigue protocol on knee joint mechanics and muscle activation during gait in young adults

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Running Head: Effect of Quadriceps Fatigue on Gait Biomechanics

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Abstract

The purpose of this study was to determine the effect of impaired quadriceps function on knee joint biomechanics and neuromuscular function during gait. Surface electromyograms, three-dimensional motion and ground reaction forces were collected during gait before and after 20 healthy adults completed a high intensity quadriceps fatigue protocol. Pattern recognition techniques were utilized to examine changes in amplitude and temporal characteristics of all gait variables. The fatigue protocol resulted in decreased knee extensor torque generation and quadriceps median power frequencies for 18 of 20 participants ($p < 0.05$). The gait data from these 18 participants was analyzed. The knee external rotation angle increased ($p < 0.05$), the net external flexion and external rotation moments decreased ($p < 0.05$), and the net external adduction moment increased ($p < 0.05$). Post-fatigue changes in periarticular muscle activation patterns were consistent with the biomechanical changes, but were not significantly altered. Even for this low demand task of walking the knee motion and loading characteristics were altered following a high intensity fatigue protocol in a manner that may place the knee joint at greater risk for joint pathology and injury.

Key Words: Quadriceps fatigue, knee biomechanics, gait, surface electromyography, knee osteoarthritis, quadriceps impairment

Effect of Quadriceps Fatigue on Knee Joint Kinematics, Kinetics and Muscle Activation during Gait in Young Adults

Introduction

Quadriceps muscles play an important role in controlling knee motion, providing stability and attenuating impact loading (Slemenda et al. 1998; Syed and Davis 2000; Lewek et al. 2002). While quadriceps impairment is often the focus of rehabilitation and prevention interventions, few studies address how impairment alters neuromuscular control and the knee mechanical loading environment. Examining direct effects of decreased quadriceps torque-generating capabilities on gait biomechanics and electromyographic patterns should provide the foundation for linking the knee mechanical environment with quadriceps impairment during a fundamental task.

Three models have been used to explore quadriceps impairment in healthy individuals: induced pain, induced effusion and induced fatigue (Torry et al. 2000; Henriksen et al. 2007; Parijat and Lockhart 2008). Reduced quadriceps activation and decreased early stance knee flexion moments have been reported using pain and effusion models (Torry et al. 2000; Henriksen et al. 2007). These models provide insight on quadriceps impairment and knee biomechanics, but are limited to drawing conclusions about impairment and pathology since pain and effusion are models of pathology. Also, alterations cannot be attributed to quadriceps impairment alone, as effusion creates physical limitations (Torry et al. 2000), and induced pain could result in pain-related compensations (Henriksen et al. 2007). Fatigue models are advantageous when examining non-pathological mechanisms as they impair muscle torque-generating capabilities, yet do not have confounding factors of pain or swelling.

Only one study examined the effect of fatigue-induced quadriceps impairment on gait kinetics and kinematics (Parijat and Lockhart 2008). After a bilateral quadriceps fatigue protocol, the only significant finding was decreased early stance knee flexion angles. This study was limited to only examining sagittal angles and moments, though quadriceps weakness has been associated with increased knee

adduction moments (Sturnieks et al. 2008). Three-dimensional kinematics and kinetics interactions are important given links between altered frontal and transverse plane mechanics and knee pathology, such as OA and anterior cruciate ligament (ACL) injury (Bonci 1999; Andriacchi et al. 2004; Olsen et al. 2004; Mundermann et al. 2005; Shimokochi and Shultz 2008). Parijat and Lockhart (2008) only examined changes in discrete variables (i.e. peaks of waveforms) whereas pattern recognition techniques such as principal component analysis (PCA) provide information on dynamic changes throughout gait (Deluzio and Astephen 2007). Finally, surface electromyograms (EMG) from lower extremity muscles were not recorded in that study (Parijat and Lockhart 2008). Lack of change in resultant joint kinetics can be due to changes in relative activation between antagonist muscle pairs (Herzog et al. 2003), so considering EMG data enhances any interpretation of joint moments. For example, individuals post-ACL injury and post-ACL reconstruction demonstrate decreased knee flexion moments, with no reduction in quadriceps activation during gait (Rudolph et al. 2001; Lewek et al. 2002). Instead the authors, using quadriceps and hamstrings EMG data, suggested that decreased knee flexion moments in this population were due to an increase in the relative contribution of the knee flexors (Rudolph et al. 2001). Interpreting moments in the context of EMG data is particularly important when using fatigue models of quadriceps impairment, as co-activation of the hamstrings has been reported as a potential mechanism for reducing extensor torques during quadriceps fatigue protocols (Psek and Cafaralli 1993). This may affect their activation levels during post-fatigue tasks: Kellis and Kouvelioti (2009) reported decreased hamstrings activity following quadriceps fatigue protocols.

This study aimed to determine the effect of impaired quadriceps function on knee biomechanics and neuromuscular function during gait. Using a fatigue protocol to induce quadriceps impairment provides a non-pathological model to study the role the quadriceps play in altering the mechanical environment of the knee related to risks of developing knee pathology (i.e. osteoarthritis) (Slemenda et al. 1998; Syed and Davis 2000) and injury (i.e. ACL) (Thomas et al. 2010). We hypothesized that a high intensity unilateral fatigue protocol would impair the torque-generating

capability of the quadriceps muscle group in healthy individuals, and alter three-dimensional knee biomechanical waveforms and periarticular muscle activation patterns during walking. Pattern recognition techniques were utilized to examine changes in amplitude and temporal characteristics of gait variables.

Methods

Participants

Twenty healthy participants (10 males, 10 females) were tested at the Dynamics of Human Motion laboratory at Dalhousie University. Participants were between 19 and 35 years to decrease the probability of asymptomatic knee OA, as the risk of knee OA increases with age (Lawrence et al. 2008). To reduce the potential for rapid recovery following the fatigue protocol they met a sedentary classification, participating in less than 30 min of moderate physical activity most days of the week (American College of Sports Medicine 2006). Participants filled out a health screening questionnaire to ensure they had no history of lower extremity orthopaedic or rheumatic pathology, surgery or trauma and were free of cardiovascular disease, neuromuscular disorders, or any condition that would adversely affect their gait or safety while participating. All participants signed an informed consent approved by the Dalhousie University Ethics Review Board.

Procedure

Participants performed gait trials before and after a quadriceps fatigue protocol (Fig. 1). To monitor muscle activity silver/silver chloride surface electrodes (0.79 mm^2 contact area, Kendall, Tyco Healthcare Group, Mansfield MA) were placed, after standard skin preparation, in a bipolar configuration (2 cm interelectrode distance) in line with muscle fibres of vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF), lateral (LH) and medial hamstrings (MH), and lateral (LG) and medial gastrocnemius (MG). A reference electrode was placed on the tibial shaft. Placements were verified by assessing EMG recordings while participants performed isolated movements to activate the different muscles

(Kendall et al. 1993; Hubley-Kozey et al. 2006). Raw EMG signals were preamplified 500× then further amplified (bandpass 10–1,000 Hz, CMRR = 115 dB (at 60 Hz), input impedence ~10 Gohm) using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta). Baseline activity was recorded while participants lay supine and relaxed (Hubley-Kozey et al. 2006).

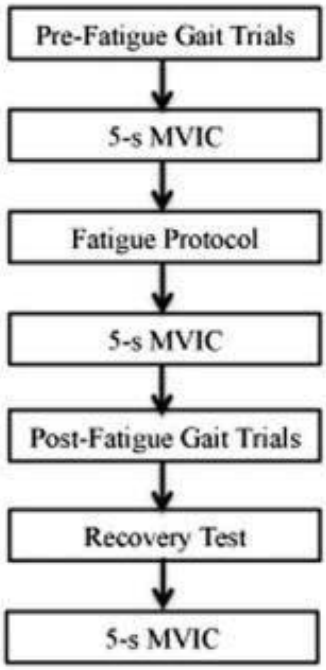

Fig. 1 Flow diagram of study protocol

Prior to the walking trials participants performed eight maximum voluntary isometric contraction (MVIC) exercises designed to elicit maximum activation of the knee flexors, extensors and plantar flexors for amplitude-normalization, with all but one performed against an isokinetic dynamometer (Cybex International Inc, MA). All exercises have been described in detail elsewhere (Hubley-Kozey et al. 2006). For each exercise, the participant was given one practice contraction, and two maximum effort contractions were recorded (for a total of 16 recorded contractions). Each contraction was held for a 3-s count. Verbal encouragement and visual feedback (torque displayed on a computer monitor) were provided. Eight different exercises

were performed, as it has been shown that no single exercise for a muscle group is sufficient to elicit maximum activation for all subjects (Rutherford et al. 2011).

Gait trials followed the normalization exercises. To monitor three-dimensional motion, 16 infrared emitting diodes (Northern Digital Inc, Waterloo ON) were placed on standard landmarks on the participant's right side. Diode triads were on the pelvis, thigh, lower leg, and foot, with individual diodes on the shoulder, greater trochanter, lateral epicondyle, and lateral malleolus. Virtual markers (right and left anterior superior iliac spines, medial epicondyle, tibial tubercle, fibular head, medial malleolus, second metatarsal, and heel) were identified in quiet standing (Landry et al. 2007).

Participants were used as their own control. Reliability testing was completed for moment and angle (n = 30) and EMG (n = 23) principal components (PCs) with waveforms showing no visible differences between a set of walking trials separated by 5 min of sitting (a time comparable to completing the fatigue protocol). ICCs from 0.93 to 0.99 were found for biomechanics PCs and from 0.88 to 0.99 for EMG PCs. Markers and electrodes were not removed between the pre-fatigue and post-fatigue gait trials.

For the gait trials participants walked along a 6-m walkway at their self-selected velocity until five successful trials were recorded. For a trial to be considered successful, the participant's right foot had to come into full contact with the force plate, with their left foot not touching the force plate. Participants had to maintain (±5%) their self-selected velocity, since EMG, kinematic and kinetic variables vary with velocity (Zeni and Higginson 2009). Velocity was measured using two infrared light timing gates controlled by LabView™ software (National Instruments Corporation, Austin TX). Segment motion was recorded at 100 Hz using an Optotrak™ motion capture system (Northern Digital Inc, Waterloo ON) synchronized with the force platform (Advanced Mechanical Technology Inc, Watertown MA) and EMG system. EMG and ground reaction forces were digitized at 2,000 Hz using the analogue data capture feature of the Optotrak™ system.

The unilateral quadriceps fatigue protocol consisted of 50 maximum effort knee extensions at 90°/s against an isokinetic dynamometer, and was based on an

endurance test described by Lindstrom et al (1995) and Larsson et al (2003). They found that 40–50 maximum effort knee extensions at 90°/s were needed to fatigue the quadriceps. The right leg was chosen for testing as no differences in torque and EMG mean power frequencies were found between the two legs in healthy participants (Lindstrom et al. 1995). Median power frequencies of the quadriceps EMG signals were determined from 5-s MVIC contractions (knee at 45º) before and after the fatigue protocol (Fig. 1). Prior to the fatigue protocol the participants performed a specific warm-up of ten knee extensions at 90°/s at 50% of their perceived maximum capability. During the protocol, verbal encouragement and visual feedback were provided: torques were displayed on a computer monitor, and participants were encouraged to exceed their previously produced torque. Recovery was assessed following the post-fatigue gait trials when participants performed five additional maximum effort dynamic knee extensions and an additional 5-s MVIC (Fig. 1).

Data Processing

Custom software (Matlab^{TM} version 7.4, the MathWorks, Natick, MA) was used to full-wave rectify, low pass filter (6 Hz, 4th order Butterworth filter), time normalize (% gait cycle), and amplitude normalize (% MVIC) EMG signals recorded during gait (Hubley-Kozey et al. 2006; Hubley-Kozey et al. 2008). Motion and force data were digitally filtered (recursive 4th order Butterworth) at filter cut-offs of 8 and 60 Hz respectively, and used to identify heel strike and toe off to define one gait cycle. Three-dimensional angles were calculated according to the joint coordinate system described by Grood and Suntay (1983). Three-dimensional net external joint moments were calculated using inverse dynamics (Costigan et al. 1992; Deluzio et al. 1993; Li et al. 1993) and represented in the joint coordinate system (Grood and Suntay 1983). Moments were amplitude-normalized to body mass, and angles and moments were time-normalized to percentage of one gait cycle (Deluzio and Astephen 2007; Landry et al. 2007; Astephen et al. 2008). For all waveforms, five walking trials for each participant were averaged to create pre and post-fatigue ensemble average profiles.

Fatigue protocol torque data were low pass filtered (5 Hz, recursive 2nd order Butterworth filter). Gravity-corrected peak torques were determined for each knee extension during the protocol (Fig. 1). The highest torques were not always produced in the first five extensions (but had occurred in all individuals within the first 10), thus pre-fatigue torque was calculated by taking the mean of five maximum torques from the first 10 contractions. Post-fatigue torque was calculated by taking the mean of torques from the last 5 contractions. Recovery torque was calculated by taking the mean of torques from the 5 recovery contractions (Gray and Chandler 1989). Raw EMG data were bandpass filtered (20–500 Hz), corrected for bias and converted to microvolts. A hanning window was used to reduce filter ringing. Power spectrums for the middle 2 s of the 5-s MVIC contractions (Fig. 1) were determined using a 4096-point fast fourier transform (Beck et al. 2008). Median power frequency has been found to be a reliable indicator of fatigue (Hedayatpour et al. 2008) and was calculated for the three quadriceps muscles as the frequency that divided the spectrum in half (Cifrek et al. 2009). Since motivation plays an important role in any study examining voluntary efforts (Al-Zahrani et al. 2009), fatigue was assessed using torque and the median power frequency of the three quadriceps EMG signals. A participant was considered to have experienced quadriceps fatigue if (i) their post-fatigue torque was lower than their pre-fatigue torque and (ii) their post-fatigue median power frequency was lower than their pre-fatigue median power frequency in at least one of the three quadriceps muscles.

Statistical Analysis

Gait waveforms were analyzed using PCA to reduce data to a limited number of principal components (PCs) explaining the majority of variability in the waveform data. The application of PCA to gait waveforms has been described in detail elsewhere (Hubley-Kozey et al. 2006; Deluzio and Astephen 2007; Hubley-Kozey et al. 2008). Briefly, the time and amplitude-normalized gait waveforms for each participant pre and post-fatigue form a data matrix for each set of waveforms for the three-dimensional kinematic and kinetic measures and the three muscle groupings (n*101, where n is the number of waveforms included in the analysis). An orthogonal transformation was applied to the data matrix, and the eigenvectors of this new matrix capture the major amplitude and shape characteristics of the original waveforms and are called PCs. The amount of

variance explained by each PC (percent trace) was calculated from the eigenvalues. Original waveform data for each participant were transformed into a set of PC *scores*, which are weighting coefficients based on how closely a waveform corresponds to a specific PC. Statistical analysis was done on these *scores*. PCs accounting for at least 90% of variance and contributing more than 1% of variance were retained for analysis (Hubley-Kozey et al. 2006; Deluzio and Astephen 2007). PCs were interpreted by comparing original waveforms for the five subjects with the highest scores to those for the five subjects with the lowest scores (Hubley-Kozey et al. 2006; Deluzio and Astephen 2007; Hubley-Kozey et al. 2008).

One-way repeated measures analysis of variance (ANOVA) models tested for significant differences in maximum knee extensor torque and quadriceps median power frequencies between three time points: pre and post-fatigue and at the recovery test. Paired Student's *t*-tests tested for pre to post-fatigue differences in velocity and PC *scores* for knee angles and moments. For quadriceps, hamstrings and gastrocnemius muscle groupings, two-factor (pre–post-fatigue, muscle) ANOVAs tested for significant pre to post-fatigue changes, differences between muscle sites and two-way interactions in PC *scores*. Alpha (α) was 0.05 for all tests. Significant interactions were further analyzed using pairwise comparisons with Bonferroni corrections. Normality (Kolmogorov-Smirnov) and equal variances (Levene's) were confirmed. All statistical analyses were completed in Minitab^{TM} version 15 (Minitab Inc, State College, PA, USA).

Results

Quadriceps Fatigue Protocol

The fatigue protocol resulted in decreased knee extensor torque and median power frequency in at least one of the quadriceps muscles, indicating induced fatigue, in all but two participants. As this study aimed to look at the effect of fatigue-induced quadriceps impairment, the two participants who did not fatigue were excluded from further statistical analysis. The demographics of the remaining participants are presented in Table 1. Gait velocity did not change between the pre and post-fatigue gait trials ($p>0.05$). The pre-fatigue velocity was 1.26 (0.21) m/s and the post-fatigue velocity was 1.23 (0.18) m/s.

All participants had decreased knee extensor torque, and all but two participants had decreased median power frequency in at least one quadriceps muscle following the fatigue protocol. As this study aimed to look at fatigue-induced quadriceps impairment, participants who did not meet the fatigue criteria (one male, one female) were excluded from further analysis. Demographics of remaining participants are in Table 1. The excluded participants were not different from those included, with their descriptive characteristics falling within one standard deviation of the means presented in Table 1. Velocity did not change between pre and post-fatigue gait trials (1.26 (0.21) to 1.23 (0.18) m/s, $p > 0.05$).

Table 1
Participant demographics ($n = 18$)

Age (years)	25 (3)
Height (m)	1.7 (0.1)
Mass (kg)	71.8 (13.3)
Body mass index (kg/m^2)	23.7 (3.3)

Data presented as mean (standard deviation)

Knee extensor torques and quadriceps median power frequencies are in Table 2. The fatigue protocol resulted in significantly decreased (40%) knee extensor torque and median power frequency for each quadriceps (14–20%). All fatigue indicators were still lower at recovery, but only VM and RF median power frequencies were significantly lower than pre-fatigue values.

Table 2
Knee extensor torque (Nm) and median power frequency (MedPF, Hz) pre and post-fatigue and after the final gait trials (recovery)

	Pre-fatigue	Post-fatigue	Recovery
Torque (Nm) (n = 18)	143.6 (29.7)	86.1 (22.97)*	135.5 (30.6)
Vastus lateralis MedPF (n = 16)	68.8 (10.9)	59.5 (6.1)*	66.4 (13.2)
Vastus medialis MedPF (n = 16)	70.4 (8.6)	60.2 (5.7)*	65.4 (9.2)*
Rectus femoris MedPF (n = 16)	89.8 (7.6)	72.4 (8.3)*	81.7 (8.5)*

Data presented as mean (standard deviation)

* Indicates a significant ($p < 0.05$) difference from pre-fatigue value. For EMG data (n = 16) one participant was missing post-fatigue data (recovery signals had reduced MedPF) and one was missing recovery data (post-fatigue had reduced MedPF) due to poor quality signals

Pre and post-fatigue ensemble average waveforms for three-dimensional knee angles are in Fig. 2. Four PCs accounted for 93.3% of variability in flexion angle waveforms. There were no significant effects of fatigue on *scores* for these PCs, indicating the flexion angle did not change post-fatigue (Fig. 2a). Waveform differences were evident during swing for the adduction angle (Fig. 2b) and throughout the gait cycle for the rotation angle post-fatigue (Fig. 2c). These observations were confirmed with the PCA results. Three PCs accounted for 93.9% of variability in adduction angle waveforms. Adduction angle PC1 *scores* significantly increased post-fatigue ($p < 0.05$), reflecting increased adduction primarily during swing as illustrated by the waveforms with high PC1 scores (Fig. 3b). Four PCs accounted for 89.9% of variability in rotation angle waveforms. Rotation angle PC1 *scores* significantly decreased post-fatigue ($p < 0.05$), indicating the tibia moved from internal rotation to external rotation relative to the femur as illustrated by the waveforms with low PC1 scores (Fig. 3d).

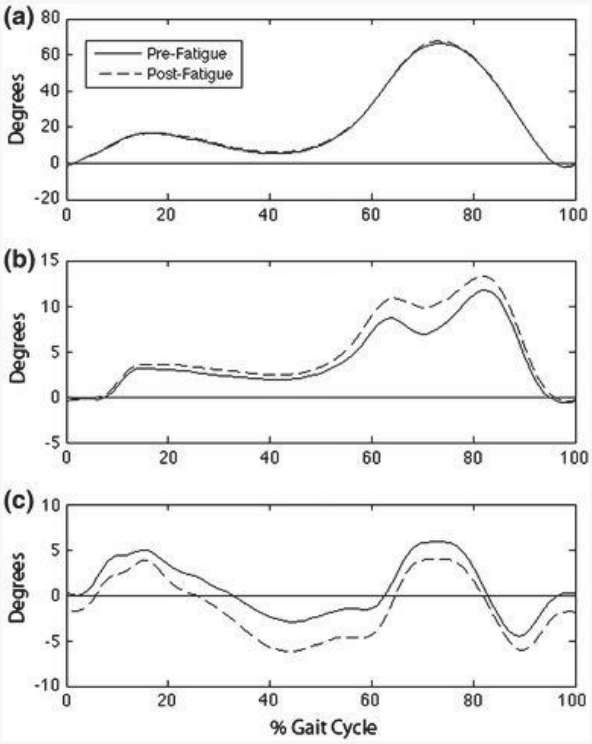

Fig. 2
Three-dimensional knee angle waveforms. The sample ensemble average waveforms (n = 18) for the pre (*solid*) and post (*dash*) quadriceps fatigue gait trials are shown for the **a** knee flexion angle (flexion positive), **b** knee adduction angle (adduction positive) and **c** knee rotation angle (tibial internal rotation positive)

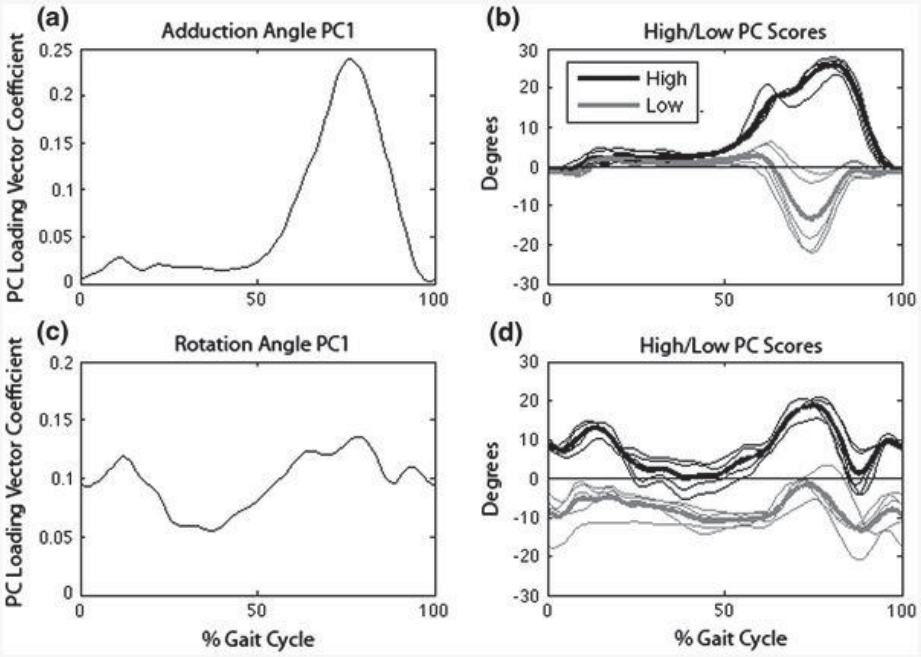

Fig. 3
Knee angle principal components. The patterns of the first principal component (PC) of the knee adduction angle and knee rotation angle are shown in the *left column*. Original waveforms for five participants having the highest (*thin black*) and five with the lowest (*thin gray*) PC1 *scores* are shown on the right (*thick lines* are means). These *high/low plots* illustrate that **b** PC1 for the knee adduction angle (accounting for 82.6% of the variance) captured primarily the angle during swing phase and **d** PC1 for the knee rotation angle (accounting for 55.9% of the variance) captured the overall magnitude of the angle over the gait cycle. Induced quadriceps fatigue was associated with higher knee adduction angle PC1 *scores* and lower knee rotation angle PC1 *scores*

Pre and post-fatigue ensemble average waveforms for three-dimensional knee moments are in Fig. 4. All moment PCs with *scores* significantly affected by fatigue are in Fig. 5 (left), with original waveforms for participants having high and low *scores* on the right. Four PCs accounted for 93.0% of variability in flexion moment waveforms. Flexion moment PC2 (Fig. 5a) captured the early stance flexion moment and late stance extension moment difference, as illustrated by the waveforms with high PC2 scores (Fig. 5b). PC2 *scores* significantly decreased post-fatigue ($p < 0.05$), indicating less difference between flexion and extension peaks. This difference was primarily the result of a decreased early stance flexion moment (Fig. 4a). To illustrate the effect, the pre-fatigue early stance peak flexion moment decreased 15.4% (0.39 (0.23) to 0.33 (0.23) Nm/kg). Six PCs accounted for 92.3% of variability in adduction moment waveforms. Adduction moment PC2 (Fig. 5c) captured the first peak and mid-to-late stance adduction

moment difference. Low-scoring waveforms had a typical pattern with first and second peaks of similar amplitude. High-scoring waveforms had high first peaks compared to mid-to-late stance values (Fig. 5d). PC2 *scores* significantly increased post-fatigue ($p < 0.05$). Thus, the post-fatigue adduction moment moved toward a pattern more similar in shape to the high score pattern depicted in Fig. 5d, in which the first peak was greater than the mid-to-late stance amplitude. Three PCs accounted for 91.4% of variability in rotation moment waveforms. Rotation moment PC1 (Fig. 5e) captured the early stance external rotation moment, illustrated by the waveforms with high PC1 scores (Fig. 5f). PC1 *scores* significantly decreased ($p < 0.05$) post-fatigue, indicating a decreased early stance external rotation moment. Again, to place this in perspective, the actual peak decrease was 11.7%.

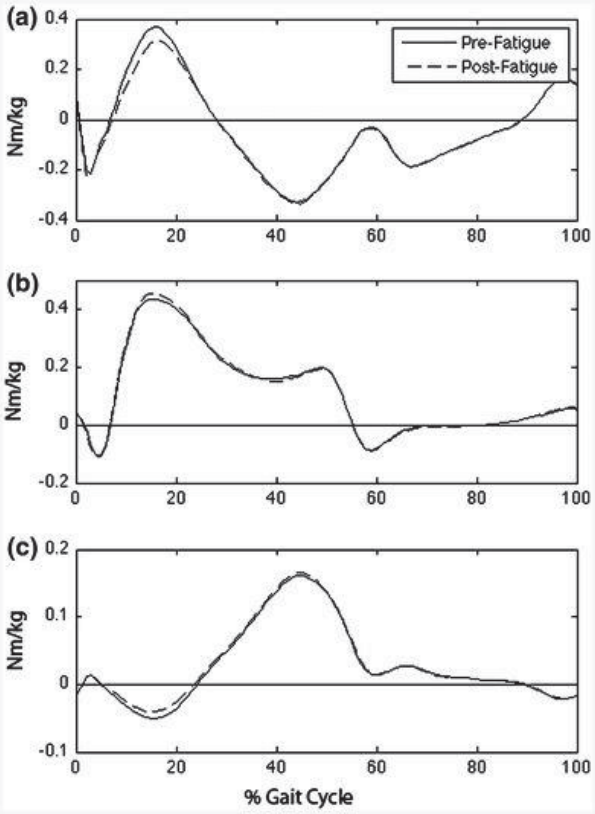

Fig. 4

Three-dimensional knee moment waveforms. The sample ensemble average waveforms (n = 18) for the pre (*solid*) and post (*dash*) quadriceps fatigue gait trials are shown for the **a** knee flexion moment (flexion positive), **b** knee adduction moment (adduction positive), and **c** knee rotation moment (internal rotation positive)

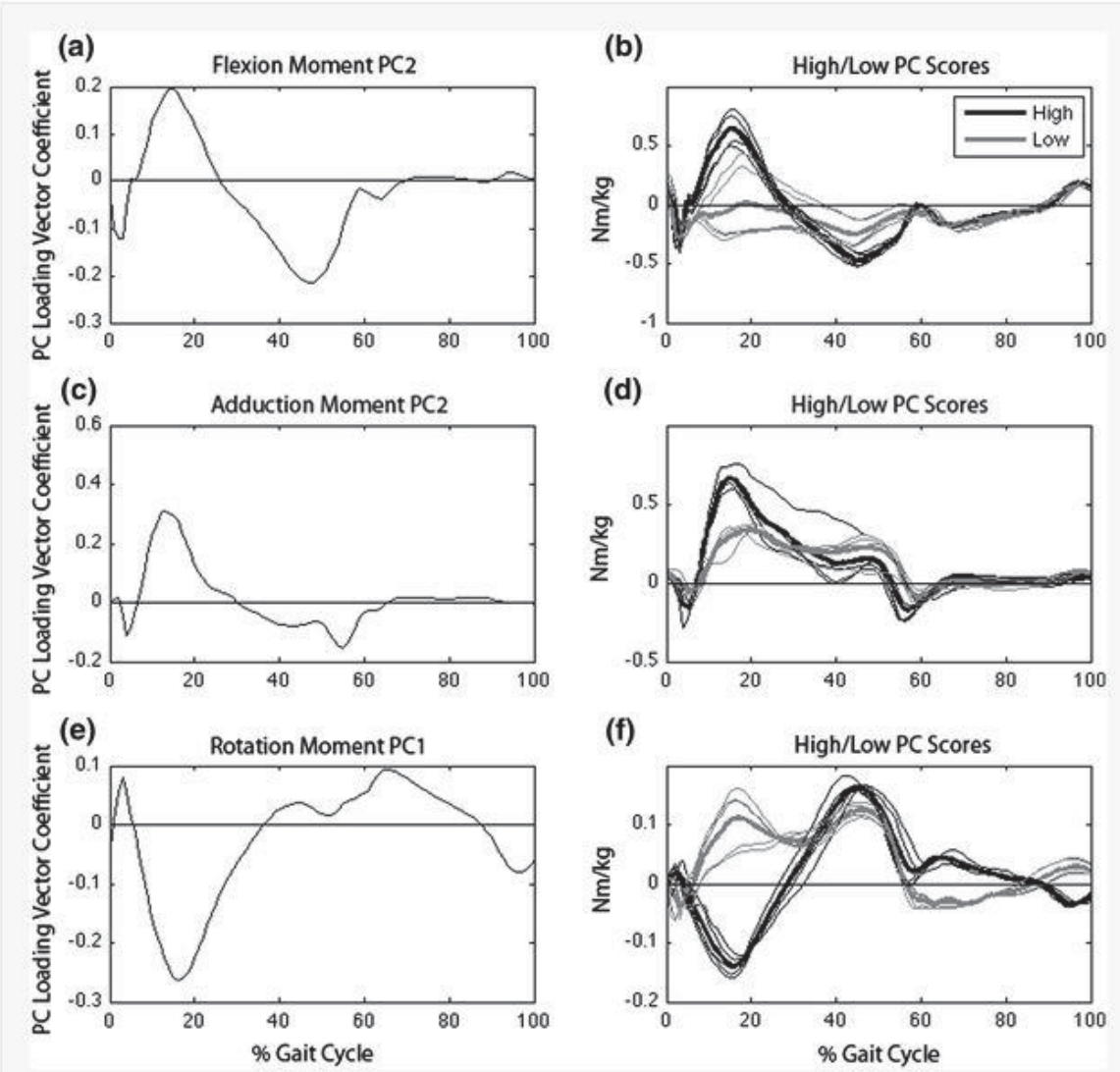

Fig. 5

Three-dimensional knee moment principal components. The patterns of the second principal component (PC) of the **a** knee flexion moment (accounting for 23.3% of the variance) and **c** knee adduction moment (accounting for 22.1% of the variance), and the first principal component of the **e** knee rotation moment (accounting for 60.1% of the variance) are shown. Original waveforms for five participants with highest (*thin black*) and five with the lowest (*thin gray*) *scores* for the depicted PCs are shown for the **b** knee flexion moment, **d** knee adduction moment and **f** knee rotation moment (*thick lines* are means). These *high/low plots* illustrate that PC2 for the knee flexion moment captured the difference between the early stance knee flexion moment and the late stance knee extension moment. PC2 for the knee adduction moment captured the difference between the first peak in the waveform and the mid-to-late stance moment. PC1 for the knee rotation moment captured the early stance external rotation moment. Induced quadriceps fatigue was associated with lower knee flexion moment PC2 *scores*, higher knee adduction moment PC2 *scores* and lower knee rotation moment PC1 *scores*

Ensemble average waveforms for all muscles (Fig. 6) except LG showed decreased activation amplitudes post-fatigue, with non-uniform changes throughout the gait cycle. PC *scores* however showed no significant pre-post test-by-muscle interactions ($p > 0.05$) or pre-post test main effects ($p > 0.05$) for any activation characteristic.

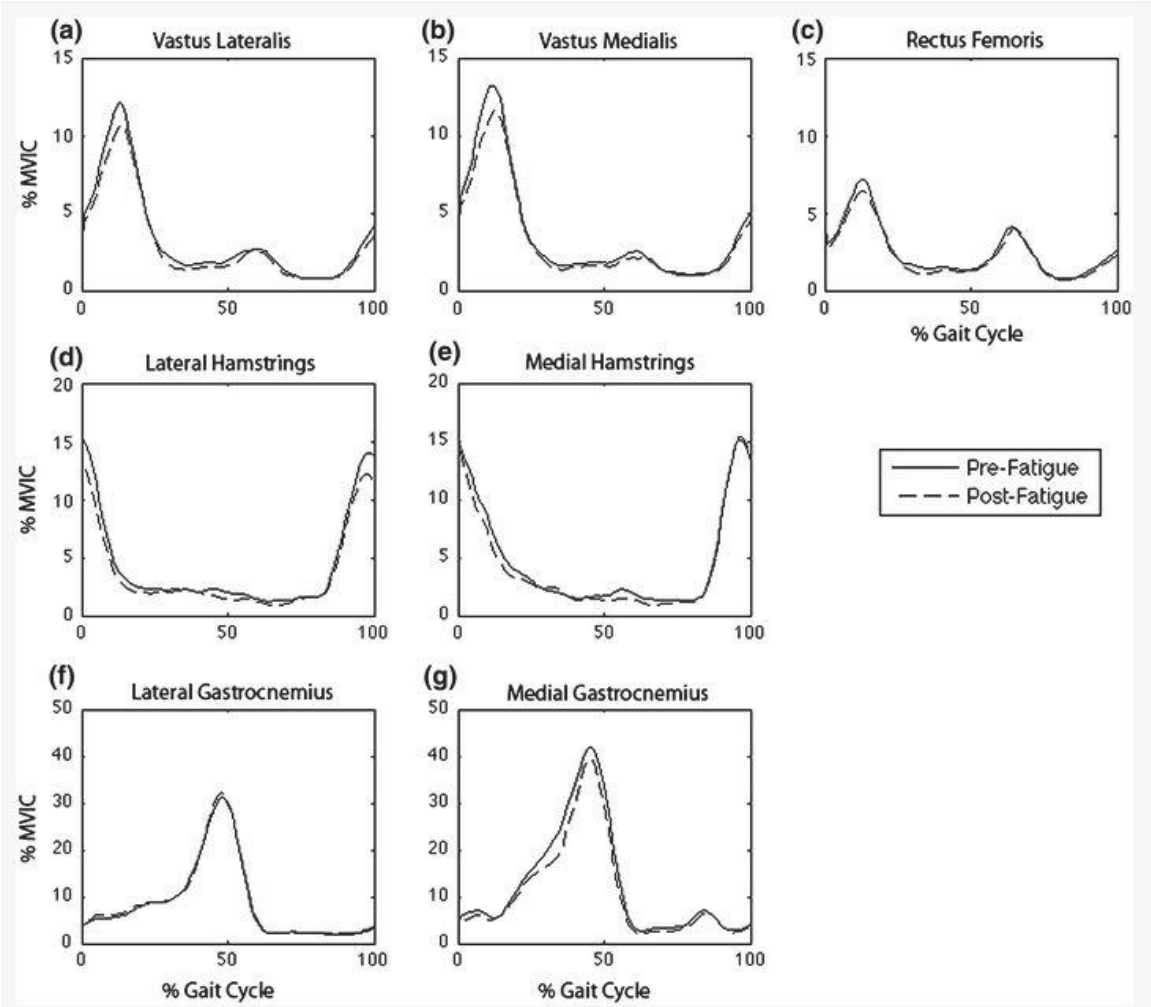

Fig. 6
Muscle activation waveforms. The sample ensemble average waveforms ($n = 18$) for the pre (*solid*) and post (*dash*) quadriceps fatigue gait trials are shown for the **a** vastus lateralis, **b** vastus medialis, **c** rectus femoris, **d** lateral and **e** medial hamstrings, and **f** lateral and **g** medial gastrocnemius muscles

Discussion

This study determined the effect of impaired quadriceps function using a high intensity fatigue model on knee biomechanics and neuromuscular activation, providing insight into alterations in the knee mechanical environment. The fatigue protocol resulted in significant impairment in torque-generating capability of the knee extensors, with torque decreasing 40% at the end of 50 contractions. A 40% deficit is clinically relevant as it is

within the range of strength deficits reported for participants with moderate to severe knee OA (Lewek et al. 2004; Hubley-Kozey et al. 2008; Petterson et al. 2008), and greater than strength deficits in a group of women who developed knee OA (Slemenda et al. 1998). The 14–20% decrease in median power frequency in three quadriceps muscles provided additional evidence that the torque decrease was due to fatigue from the exercise protocol.

Induced quadriceps impairment resulted in kinematic and kinetic changes at the knee. In contrast to previous findings (Parijat and Lockhart 2008), no knee flexion angle changes were seen, but the knee flexion moment waveform was altered post-fatigue. The lower early stance moment is consistent with Parijat and Lockhart (2008), although their finding was not statistically significant following a bilateral fatigue protocol. Their non-significant finding may be explained by a lower intensity fatigue protocol (i.e 70% MVIC) and that walking velocity differed between the fatigue and no fatigue conditions. The also provided no physiological indicator of fatigue induced by the protocol (i.e. no torque reductions or EMG measures reported). Furthermore the sedentary classification of our group could have decreased the quadriceps recovery post-fatigue, and because all subjects had to meet a sedentary criterion, our group may be more homogeneous. No indication of participant fitness was provided in the Parijat study and the torque values reported (200–700 Nm) for their pilot subjects were very high compared to the present study. The lower early stance flexion moment is consistent with impairments induced using pain (Henriksen et al. 2007) and effusion (Torry et al. 2000) models. Decreased knee flexion moments have been suggested as an indication of decreased eccentric quadriceps work in early stance, with an associated decreased ability to attenuate impact loads (Syed and Davis 2000; Lewek et al. 2004), although alterations related to antagonist co-activation have also been presented (Rudolph et al. 2001).

Changes in frontal plane mechanics were seen post-fatigue. The knee adduction angle indicates varus alignment, and while small early-stance increases were seen, consistent with increased varus thrust during weight acceptance, the greatest increase occurred in swing when the knee was unloaded. The increased knee adduction moment in early-stance was consistent in timing with the adduction angle change during early-stance. Increased knee adduction moments indicate higher medial tibiofemoral compartment

loading (Mundermann et al. 2005). While only a small difference was apparent in the initial peak, the post-fatigue alterations (PC2) captured the difference between the early stance peak and mid-to-late stance amplitudes with higher early stance peaks implicated in knee OA severity (Mundermann et al. 2005). The results suggest alterations to the typical waveform with the post-fatigue pattern moving toward a higher early stance load compared to mid-to-late stance as illustrated by the high scores in Fig. 5d. In terms of rotation, the tibia became more externally rotated on the femur post-fatigue and the early stance external rotation moment was decreased. While the changes were not as apparent as with the sagittal plane moments, and the clinical significance is unknown, the results indicate that quadriceps impairment did alter non-sagittal mechanics. To our knowledge these are the first data to examine the frontal and transverse planes with quadriceps fatigue.

The biomechanical changes in these healthy, young participants are similar to those reported for individuals with moderate knee OA. A decrease of approximately 20% in the early stance knee flexion moment has been reported in those with moderate knee OA, compared to asymptomatic controls (Astephen et al. 2008), just slightly higher than the 15% decrease found in this study. This, along with knee adduction moment alterations creates an altered loading environment for the joint. Less well studied are rotational changes, although changes in knee rotation are hypothesized to initiate the degenerative process of OA by shifting loading to unconditioned areas of cartilage, even without previous joint pathology (Andriacchi et al. 2004; Andriacchi et al. 2006). Landry et al (2007) reported decreased external rotation moments in those with moderate OA, but the magnitude of the decrease was higher than found in this study. At this point, how rotational changes affect the joint is unclear, but the results suggest that normal rotational mechanics related to the knee screw-home mechanism were altered post-fatigue.

Altered biomechanics have implications related to knee OA, but also for knee injury risk. Altered knee rotation has been proposed as a mechanism of ACL injury (Bonci 1999; Olsen et al. 2004; Shimokochi and Shultz 2008). Post-fatigue the tibia became more externally rotated with respect to the femur. Tibia external rotation is implicated in ACL injury through impingement of the ligament against the femoral condyle (Olsen et al. 2004; Shimokochi and Shultz 2008), or through increased strain on the ligament

(Bonci 1999). Thus these preliminary findings suggest a potential mechanism for increased risk of ACL injury post-fatigue, particularly if an individual was to engage in activities that involve high mechanical strain on the knee joint while in a fatigued state.

Induced fatigue resulted in amplitude decreases in all periarticular knee muscles studied, excepting LG. These decreases did not reach statistical significance as found with effusion and pain models (Torry et al. 2000; Henriksen et al. 2007). This is likely because these were healthy, young participants working at very low percentages of maximum voluntary capacity during the walking trials (Fig. 6). Also there is inherently more variability in EMG data and a power analysis indicated we would need a large sample size ($n = 58$) to detect a 15% decrease in quadriceps amplitudes (the magnitude of the change seen for the knee flexor moment). These decreases in activity, though not significant, are implicated in the biomechanical changes and require further investigation. The decreased quadriceps and hamstrings activity is consistent with the decreased knee flexion moment and may also help explain the altered knee adduction moment. Sturnieks et al. (2008) found higher knee adduction moments in individuals with decreased quadriceps strength. Decreased hamstrings activity after quadriceps fatigue has previously been reported for a jumping task, and is referred to as an antagonist inhibition strategy (Kellis and Kouvelioti 2009). This strategy is suggested to be compensatory, increasing knee mechanical efficiency (Padua et al. 2006). The altered muscle force generating patterns may explain the rotational mechanics changes since stabilizing forces would presumably be reduced with fatigue and the associated decrease in activation amplitudes.

The findings must be interpreted within the context of the limitations. Caution must be exercised when interpreting non-sagittal knee motions obtained from gait analysis to ensure that observed motions are not due to kinematic crosstalk. Piazza and Cavanagh (2000) demonstrated that measurements of knee rotation are influenced by errors in location of the flexion axis. However, crosstalk predominantly occurred at large knee flexion angles with little to no erroneous knee rotation seen from 0–15° of flexion. Although some kinematic crosstalk may have occurred in this study, it would likely have occurred in swing, when greater degrees of flexion were reached. Changes in knee rotation during the loading phase (around 15° of knee flexion) are unlikely to be due only

to crosstalk. In addition, changes in knee adduction and rotation angles were not uniform throughout the gait cycle and thus do not support a systematic effect.

By the time participants finished the post-fatigue gait trials, some fatigue parameters (i.e. quadriceps torque and vastus lateralis median power frequency), while still lower, were not significantly different from baseline. Ensuring long-lasting fatigue from a short duration protocol was difficult. To minimize the effects of recovery, participants started the post-fatigue gait trials within 2 min of completing the fatigue protocol and this is well within the 10-min time frame suggested by Parijat and Lockhart (2008). Despite apparent recovery in some parameters, vastus medialis and rectus femoris median power frequencies remained significantly different from baseline values, and kinematic and kinetic changes were still observed. If fatigue had lasted longer, changes may have been even more evident. This is a challenge, as high intensity fatigue effects may not be representative of fatigue induced through low-level activation seen in activities of daily living. The present fatigue protocol is more representative of that occurring in high intensity athletic activities and thus cannot necessarily be generalized. However, what the results do illustrate is that induced impairment from a non-pathological mechanism resulted in alterations to biomechanical variables that are important to the mechanical loading environment of the knee. Subsequently they provide evidence of a cause-effect relationship. Future studies of quadriceps fatigue should examine the effect on performing more demanding post-fatigue tasks that would require higher amplitudes of muscle activation to better understand injury, whereas lower intensity protocols may better reflect knee OA initiation/progression mechanisms. The implication for practice relates to the threshold for reserve and ensuring that sufficient strength and endurance are maintained to provide a positive mechanical environment.

In conclusion, this study showed that a high intensity unilateral fatigue protocol did impair the torque-generating capability of the quadriceps muscle group in healthy individuals and that post-fatigue changes in knee motion and loading characteristics during walking were consistent with alterations to the typical knee biomechanics patterns. The changes in the mechanical environment are consistent with placing the knee at greater risk for injury (such as ACL tears) or for degenerative changes. While changes in

periarticular muscles were not statistically significant, the decreased quadriceps and hamstrings activations were consistent with biomechanical changes observed.

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